Abstract Nr: 5

Paper title: **LACK OF EVIDENCE FOR A MAJOR ROLE OF VEGF AND PlGF IN EDEMA FORMATION AND EXTRAVASATION IN NASAL POLYP TISSUE**

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Background: Edema represents a major feature of nasal polyp (NP) tissue and may be linked to the presence of vascular endothelial growth factor (VEGF) and placental growth factor (PlGF). Both are involved in vascular permeability and edema formation in diseases like cancer-associated retinopathy, macular edema and cutaneous inflammation. We evaluated the expression of VEGF, PlGF and their receptors in NP tissue compared to healthy nasal mucosa under baseline and inflammatory conditions and correlated these factors with markers of edema.

Methods: Nasal mucosa was taken from healthy donors (n=20) and polyp tissue from NP patients (n=18) and wet/dry ratios were determined. Expression levels of VEGF, PlGF and receptors VEGFR1 and VEGFR2 were measured by real-time RT-PCR. Protein levels of VEGF, PlGF and albumin were measured in the supernatants of homogenized tissue by ELISA. $1x10^6$ cells of suspended nasal mucosa (n=12) and polyp tissue (n=9) were stimulated with IL-1β (10ng/ml) and TNFα (10ng/ml) for 24h. Expression of VEGF, PlGF and receptors was determined by ELISA and real-time RT-PCR.

Results: Albumin levels and wet/dry ratios were higher in NP tissue compared to healthy nasal mucosa. VEGF expression was lower in NP tissue on both protein (87.03±11.49 vs. 55.55±10.42 pg/ml, P=0.034) and mRNA level (11.75±1.994 vs. 6.276±1.56, P=0.0229) whereas PlGF showed similar levels. Only inhibitory VEGFR1 was up-regulated in NP tissue (0.9412±0.2218 vs. 9.894±2.41, P=0.0003) whereas VEGFR2 was unaltered. IL-1β induced higher expression of VEGF and PlGF in control tissue and significantly reduced expression of VEGFR2, but had no effect on NP tissue. Expression levels of VEGF, PlGF and their receptors did not correlate with edema markers.

Conclusion: Based on the differential expression patterns and *in vitro* induction of VEGF, PlGF and their receptors, our data point towards a lack of major contribution of these angiogenic factors to the edema formation in NP disease.